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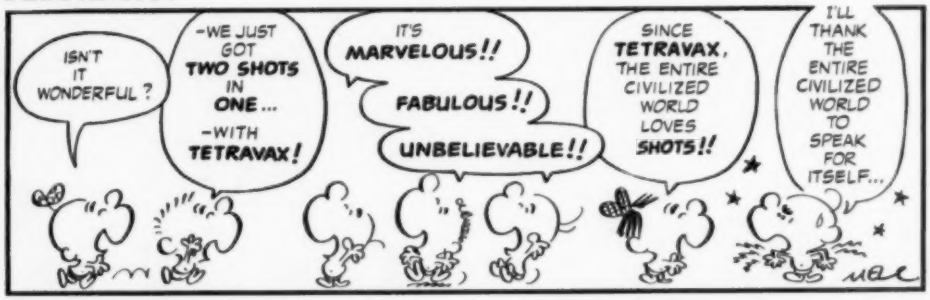
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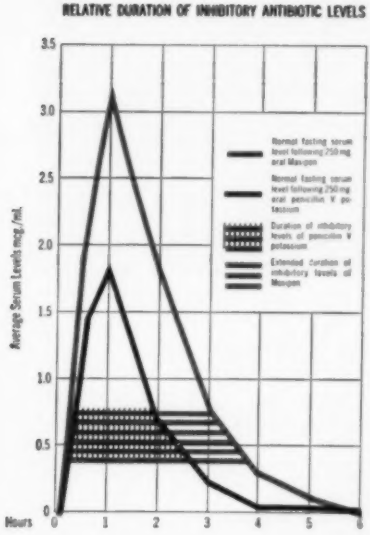

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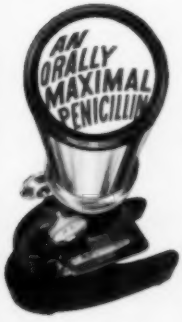

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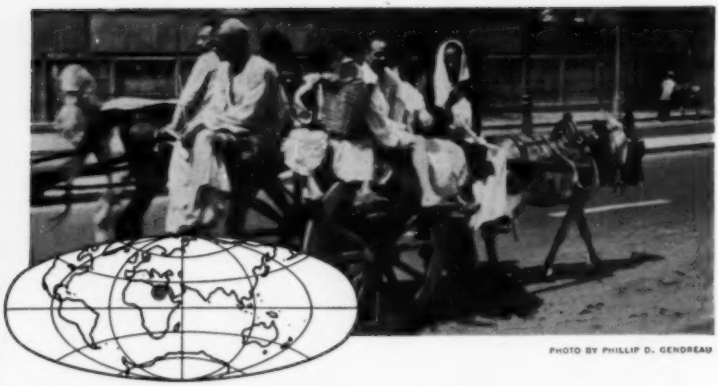

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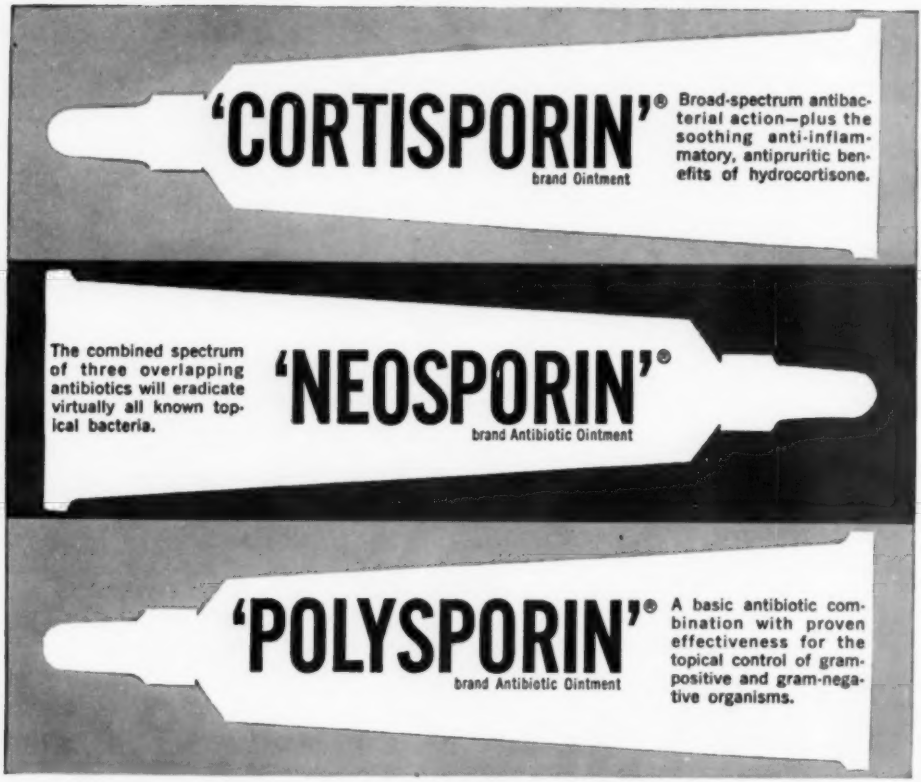
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guest editorials

Recent Concepts in Physiology and Pathology of the Newborn

PHILIP SCHWARTZ, M.D.*
Pennsylvania

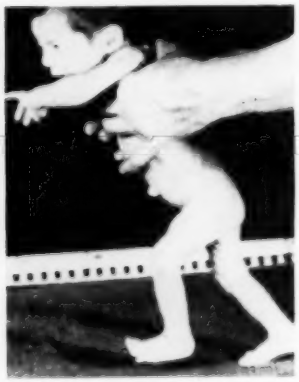
Knowledge and theory concerning physiology and clinical pathology of the newborn hitherto have been influenced by the conviction that the organism is immature at the time of delivery and that its undeveloped functions quite obviously differ from the optimal efficiency of an adult. An infant at birth does not speak, nor does he walk. He seems to be a defenseless object of the environment. His hearing is defective or completely lacking. He opens his eyes, but does not perceive. His movements are accidental and inappropriate—at any rate, aimless. Some of the reflexes produced in response to certain stimuli are due to the underdevelopment of the nervous system, reminiscent of reactions of lower vertebrates (Peiper). The Babinski phenomenon, signifying serious disease in adults, reflects the incomplete myelinization of the pyramidal tract. Athetotic and choreotic movements of the graceful small fingers witness that the extrapyramidal system, as yet, is incapable of exerting its normal inhibitory effect. ("The newborn baby is a pallidum creature." Peiper). No wonder that some of the babies display spasms: it is due to the physiologic immaturity of the brain. Icterus occurs because the liver, too, is not developed. Immature red and white blood cells swarm in the circulating blood; also, regulation of body temperature is unstable with manifestations of capricious fluctuations—hypothermia or hyperthermia.

It is true, these patterns are not identical in all neonates. However, may not these variations, too, indicate the embryologically determined incompleteness of the organs and the imperfect coordination of their function?

*Chief, Dept. of Pathology, Warren State Hospital, Warren, Pa.; Prof. of Gen. Path. and Path. Anat., Univ. Frankfurt-M., Germany; Former Director, Dept. Path. at the Univ. Istanbul, Turkey.

To be sure, surprising capabilities of normal newborn babies have often been reported. Also, the physiologic interpretation of certain phenomena, such as the Babinski reflex or spasms, has been questioned quite often. Furthermore, in connection with pathoanatomic observations concerning parturitional cerebral lesions, extensively reported during the early 20's by Ylppö, Holland, Schwartz, Wohlwill, Siegmund, and many others, new clinical signs were discovered, such as the spontaneous nystagmus (Voss, Berberich, Stern) and disturbances in the caloric and rotatoric irritability of the vestibular apparatus (Voss, Berberich), the morbid significance of which seemed obvious. In spite of all these facts, the conviction still prevailed that the behavior of newborn babies is primarily conditioned by the immaturity of the nervous system and of the organs in general.

However, we are of the impression that currently these traditional concepts are being subjected to a gradual transformation which may eventually lead to consideration of the organism of a normal newborn baby as physiologically adequate for the age, comparable in many of its accomplishments and manifestations with those of healthy adults.



Automatic "walking" of a newborn baby (Peiper)

Newborn baby climbing stairs (Peiper)

One of the most striking observations, *i.e., automatic walking movements of newborn babies,* was made by A. Peiper in 1928. (Fig. *a* and *b*) André-Thomas, rediscovering the same phenomenon in 1946, maintained that the automatic gait of the neonate corresponds to the instinctive ambulation of newborn animals, with the difference that in man it has to be induced, whereas it is spon-

taneous in animals. Another important fact disclosed by André-Thomas was that a normal newborn is neither astasic nor abasic, and that astasia (= inability to stand) and abasia (= inability to produce the mechanism of gait) in the newborn are of morbid nature, or at any rate, abnormal phenomena.

From the pathological viewpoint, Peiper, in the second edition of his excellent book "Particularities of Cerebral Function in Children," 1956, reported on his own extensive and varied observations regarding neonatal neurophysiology and summarized exhaustively the literature. His associate, Prechtl, endeavored to benefit in clinical pathology of the newborn from these studies. Prechtl's most impressive results are as follows: Out of 218 full-term babies with histories of complications at delivery, 126 (58 per cent) showed morbid aberrations—reflex lateralization, hypokinesis, hyperirritability with trembling movements, hypo- or hypertonia, absence of Moro's reaction or of oral reflexes, etc., and epileptic manifestations. According to Prechtl, neurological abnormalities in neonates have a prognostic value in regard to the later emergence of behavior problems, such as sleep disturbances, restlessness, lability of mood, anger, irritability, poor concentration. (See also Knobloch and associates' "Syndrome of minimal cerebral damage in infancy.")

Of further great value are André-Thomas' studies on the neurologic and psycho-affective behavior of normal and diseased newborns. Whereas Peiper—impressed by philosophical and phylogenetical doctrines—tried to minimize the significance of parturitional influences in explanation of discrepancies concerning neurological and psycho-affective phenomena occurring in newborn babies, André-Thomas and his associates were perhaps more practical in evaluation of their results. Polani based his systematic investigations on André-Thomas' studies.

C. A. Smith's "Physiology of the Newborn Infant" (1959) can be considered to be an introduction to a new phase of newborn pathology, in which methods and points of view of internal medicine will increasingly predominate. Indeed, Smith's book is presently no more than the first attempt to collect observations and opinions—mostly of American and English authors—concerning some selected subjects. Also, Smith's review is perhaps too strongly colored by the conviction that the organs of the newborn are immature, and therefore function differently than in adults. Phenomena connected with parturitional lesions of the brain and peripheral regulatory apparatus, of the blood, liver, kidneys and

adrenals, are either not discussed at all, or only insufficiently taken into consideration. Nevertheless, Smith's work complements studies carried out by Peiper, André-Thomas, Knobloch, F. K. Graham and their associates, and is of great value for future research on normal and morbid conditions in the newborn, with especial regard to the influence of parturitional injuries.

It should be productive to investigate both the anatomical structure and the physiological function of the newborn with the same standards accepted as normal in adults. Thus, every deviation from these norms could be considered in the newborn too, as a possibly pathologic phenomenon. This attitude, of course, may lead to mistakes. Even errors originating from this practice, however, might have productive aspects. The traditional concept, which regards as "physiological" the most amazing divergencies in the composition of tissues and of the blood in the newborn, has caused as much confusion as the concept that the most astonishing functional variations are normal phenomena of an "immature" organism. The physiology and clinical pathology of the newborn should be reconsidered. Thereby, a wide and fruitful field for systematic research might be opened.

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Mesenteric Cysts and Chylous Ascites

Robert H. Whittlesey, M.D., F.A.C.S.
Guenther H. Heidorn, M.D., F.A.C.C.
Wellington B. Huntley, M.D.
North Dakota

Five years ago Ingram, Riker, and Whittlesey published a report of chylous ascites in childhood[10]. In discussing the etiology of chylous effusion into the body cavities, we failed to mention that accumulation of chyle within the peritoneal cavity which is encountered with congenital mesenteric cysts. Recent surgical encounter with a mesenteric cyst associated with chylous ascites in a seven year old female has prompted the present communication.

That these lesions are uncommonly encountered is immediately apparent from a review of pertinent literature. Presumably Benivieni gave the first description of such a lesion in 1507, but it was almost 350 years later that a mesenteric cyst was adequately describ by Rokitansky[7]. About ten years ago it was stated that some 600 cases had been recorded in the world medical literature, and that slightly less than half of these had been described as being of the chylous variety. Vaughn is the only other author we have been able to find, who has described a chylous mesenteric cyst with calcification similar to that to be described in the following report[9]. Further evidence of the rarity of this lesion is gained from the paper of of Beahrs, which described only seven cases encountered in over 1,000,000 patients from the Mayo Clinic[1]. Slocum found six cases in the records of the Massachusetts General Hospital in a 25 year period[8]. One of the most interesting case reports is that of Kron which described a chylous mesenteric cyst causing volvulus of a malrotated intestine[5]. We have been unable to find any article dealing with this subject in the national medical journals of United States published within the last five years, although there have been numerous case reports in the medical literature from England and the continent during this period.[2]

In a discussion of isolated mesenteric cysts, Gross cited his experience with 12 cases[3]. He noted that such cysts may arise in any portion of the mesentery or of the mesacolon, sometimes reaching

enormous proportions. In his experience, the fluid within the cysts is either serous and colorless, or chylous, in which case it is whitish or yellowish. In another study of chylous cysts of the mesentery in children, Ravitch and Handelsman found that such lesions are rare abdominal tumers[4]. These authors felt that the lesions represent either true lymphagiomata of the mesentery, or are the result of lymphatic obstruction. They quoted their experience with four cases of true, chylous mesenteric cysts. In each instance, pain was the prominent feature of the illness which brought the patient to seek medical attention.

CASE REPORT

M.A.H., a seven year old white female, was referred to the clinic because of recurrent abdominal distention, fever, leukocytosis, and abdominal pain with vomiting. Her present difficulty had actually begun four years previously, at the age of three, when the first episode of abdominal distention was noted. After some initial increase, the distention regressed, but returned the following year, when it increased to the point that a paracentesis was performed. 1000 ml. of chylous fluid were obtained at that time. She was then referred to a university hospital, where studies led to the conclusion that she was the victim of chylous ascites on a congenital basis, and of a relatively mild degree. She was placed on a high caloric, low fat diet, and returned to her family physician. The following year another paracentesis was performed, but at this time only a small amount of serosanguineous fluid was obtained. Six months later another paracentesis produced 250 ml. of milky fluid. She was then not seen by another physician until 18 months later, at which time the abdomen was enormously distended and paracentesis is said to have produced 4000 ml. of chyle. At this time, she complained of abdominal cramps, and diarrhea and low grade fever were recorded. Following this final paracentesis she was seen at this clinic.

At the time she was first seen, physical examination revealed a thin, debilitated and chronically ill young, white female. Examination of the chest revealed marked elevation of the diaphragms bilaterally. The abdomen was markedly protuberant and tightly distended. There was pain on mild palpation. The abdomen had a definitely doughy consistency. Bowel sounds could not be heard. Neither the liver nor the spleen could be felt. A fluid wave was present. The laboratory work revealed a normal urinalysis. The corrected sedimentation rate was 36; hematocrit 35 vol. %; hemo-

globin 10 gms.; white blood count 18,000 with 90% segmented neutrophils; blood sugar 75 mgs. %; BUN 10 mgs. %; Prothrombin time 56%; total bilirubin 0.5 mgs. %; and 1+ cephalin flocculation; CO_2 21.4 milliequivalents per litre; chlorides 94.5 milliequivalents per litre, sodium 129 milliequivalents per litre; potassium 5.0 milliequivalents per litre. Total proteins were 5.5 grams with 3.3 grams % albumin, 2.2 grams % globulin. An x-ray of the chest showed the diaphragms to be markedly elevated (Fig. 1). An abdominal x-ray showed the intestines to be displaced into the left upper quadrant (Fig. 2). In the region of the right ileum, there was

Figures 1 & 2. Roentgenograms reveal an homogeneous opacity of considerable density filling out almost the entire abdomen. Diaphragms are both markedly elevated. The intestines are displaced into the left upper quadrant. Over the right ileum, there is a calcareous shadow faintly "resembling a phalanx or metacarpal bone".

a calcareous shadow faintly "resembling a phalanx or metacarpal bone". It was felt that the findings were suspicious of a dermoid cyst. Intravenous pyelography was performed (Fig. 3). There was a double kidney on the left with two separate ureters which merged at the level of the crest of the ileum. The right kidney was displaced upward. The right ureter was markedly displaced medially in its upper third and at its distal end. An electrocardiogram showed no abnormality. Multiple paracenteses were then carried out through various sites on the abdominal wall. The varying fluid characteristics, from clear yellow to turbid white, aroused the suspicion that multiple cysts were involved. The patient was taken to surgery.

The abdomen was entered by a low transverse incision. An enormous soft, gelatinous, cystic mass was immediately encountered. Small bowel ran through a groove in the center of this mass. It was estimated that the lower two-thirds of small intestine below the

ligament of Treitz was so intimately involved with the cysts, circulationwise, as to necessitate resection. An aseptic, end-end anastomosis of small intestine was accomplished after this resection and removal of the specimen. The patient tolerated these procedures well.

Figure 3. Intravenous pyelogram demonstrates a double kidney on the left with two separate ureters merging at the level of the crest of the ileum. The right kidney is displaced upward and the right ureter is displaced medially in its upper third and at its distal end.

The specimen presented to the pathologist consisted of small bowel and mesenteric tumor. There was a large multiloculated cystic mass measuring 26 x 26 x 7 cms. Attached to one side of this mass was a 23 cm. length of small intestine. This small bowel averaged 3.5 cms. in circumference and was essentially grossly unremarkable, except that it was firmly adherent to the cystic tumor for a distance of approximately 8 cms. When the bowel was opened, there were seen to be no communications between the lumen of the bowel and the lumina of the various cysts. The specimen was searched for evidence of ossification or calcification but none was found. The locules which composed the mesenteric cyst measured from a few millimeters to eight centimeters in diameter. Microscopically, sections from this specimen revealed portions of small bowel

involved with edema, hemorrhage, hyperemia, and chronic inflammation. The cyst wall was composed of orderly layers made up of spindle-shaped cells resembling fibrocytes (Fig. 4). In many areas there were multiple small cystic cavities, somewhat reminiscent of lymphatic channels. There was no evidence of malignancy in any of the sections examined.

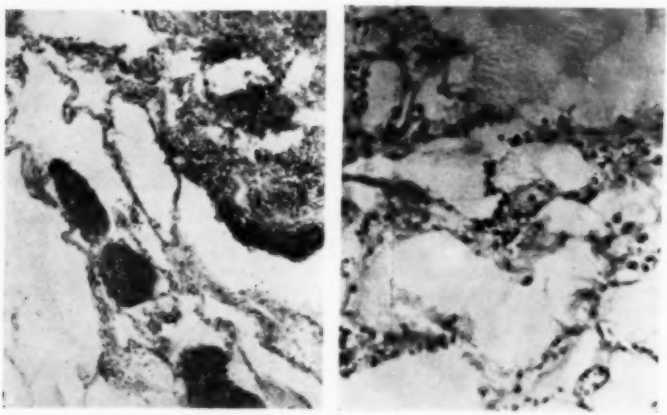

Figure 4. Microscopic section reveals cyst wall to be composed of orderly layers of spindle-shaped cells resembling fibrocytes. Multiple small cystic cavities are somewhat reminiscent of lymphatic channels. These thin-walled cysts are identical to the walls of large cystic locules. Within the wall of the cyst and attached to it is normal adult fat. Infiltrating the fat and fibrous tissue are moderately large numbers of lymphocytes, a few plasma cells, and an occasional neutrophil.

The patient's post-operative course was uneventful, although she was kept in the hospital approximately three weeks following surgery, mainly for the purpose of improving her nutrition. She has continued to do well at home in the months following her discharge from the hospital.

DISCUSSION

Mesenteric cysts in childhood are uncommon enough that each case is of interest to the clinician. Presumably, these lesions represent congenital abnormalities developing from misplaced bits of lymphatic tissue which proliferate and accumulate fluid because they do not possess communications which allow them to drain properly into the remainder of the lymphatic system. Most commonly they

are found in the mesentery of the small intestine, but may occasionally appear in the transverse mesocolon or mesosigmoid.

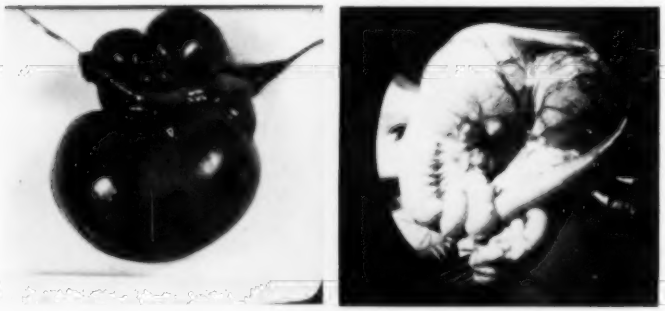

Figure 5 & 6. Multiple mesenteric cysts from two other cases which closely resemble the position of the cysts found at laparotomy in case presented, except that those illustrated are larger and there is less variation in the size of the multiple cystic cavities. In both cases the cysts are immediately adjacent to the small intestine, the circulation of which is closely intertwined with the blood supply of the cystic cavities. In both cases, removal of the cysts necessitated resection of a portion of small intestine.

The nature of the fluid within the cystic cavities has been analyzed by Messer[6]. Fluid from serous mesenteric cysts has approximately the same electrolyte concentrations as does other extracellular fluid. There is, however, 68 mgs. % of cholesterol. Examination of chylous fluid from mesenteric cysts showed chlorides 108 millequivalents per litre; non-protein nitrogen 29 mgs. %; total protein 2.1 grams %; and fat 1%.

These tumors should be pathologically distinguished from enteric cysts, which actually represent duplications of the gastrointestinal tract, but which are comonly mistaken for this lesion. The cysts may be found at any age during infancy and childhood. Characteristically, they present as a mass in the abdomen or as a slowly enlarging abdomen. They are frequently associated with bouts of abdominal pain, at times associated with vomiting. There may be reduced appetite and failure to gain weight, and in occasional patients, symptoms of acute intestinal obstruction present. Once the diagnosis becomes clear the treatment is surgical. Ideally, the cyst may be dissected out from the mesentery, but if the mass is unduly adherent to the intestine, it must be excised along with a portion of the intestine as in the case presented. Marsupialization, as was formerly done, has no place in present-day practice[8]. Of particular

importance to the clinician dealing with these children is the need for an accurate diagnosis. In the case recorded much time was lost and the well-being of the patient jeopardized through inability to correctly interpret the physical abnormality. To the clinician attuned to the fact that the discovery of chyle within the abdominal cavity may represent encysted fluid as well as free ascitic fluid, the need for careful diagnostic x-ray studies will be apparent.

SUMMARY

The case of a seven year old white female who harbored a congenital mesenteric cyst for at least four years is recorded. Chyle obtained by abdominal paracentesis originally led to the incorrect conclusion that medical therapy was indicated. Recurrence of the ascites finally resulted in the correct establishment of diagnosis and the more appropriate surgical extraction of the lesion. The association of mesenteric cysts with chylous ascites must therefore be appended to the list of etiological mechanisms gathered by the author in his previous communication.

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Pityriasis Rosea in a Three Month Old Infant

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California

In 1860 Gilbert first described pityriasis rosea. Since then many cases have been reported and now the literature is replete with reports of this disease entity. Some investigators feel that pityriasis rosea is more common than it was 40 years ago.

In 1940 Niles and Klumpp made an excellent review of the literature and reported two hundred nineteen cases. They reported the youngest patient with the disease. This was a 7 month old patient of Crocker. No report of any patient younger than 7 months has been found after a thorough review of the literature.

This case is presented because it is felt that this 3 month old infant is the youngest patient ever reported with pityriasis rosea.

CASE REPORT

E. E. C. a 3 month old Negro female was born August 5, 1959. At birth she weighed 4 pounds and 8 ounces. She had done well since birth. On September 15, 1959, she was seen in my office as a well baby. She was seen in my office the second time on November 6, 1959. At this time she had an upper respiratory infection for one week. This was mild and the only treatment given was clopane hydrochloride nose drops 0.5%. She also had a rash that caused the mother great concern. On November 2, 1959, the mother had noticed a single, small, ringworm-like lesion on the right shoulder which subsequently progressed to depigmentation. Next two lesions appeared on the abdomen followed by a lesion on the chest. The lesions then appeared on the back. The mother stated that once these lesions began they enlarged centrifugally.

Physical examination revealed a well developed, well nourished 3 month old Negro female. A buff-colored, scaly, circinate, depigmentated eruption was seen on the chest, abdomen, and face. These

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scales were fine and crinkly. Pityriasis rosea was considered at this time as well as ringworm. The marginal accentuation of the lesions resembled the lesions of ringworm although the crinkly, thin scales were more characteristic of pityriasis rosea. It was felt that Vioform ointment would be helpful since its antifungal properties would be utilized if the lesions were ringworm.

On November 10, 1959, the child was seen again. No signs of infection were present. The child seemed well and happy. A few more lesions had appeared on the face. Vioform ointment was continued although it was felt that the lesions were not influenced by use.

On November 19, 1959, the child was seen in consultation by Dr. Robert L. Day, of Bakersfield, California, a dermatologist. Dr. Day's primary diagnosis was pityriasis rosea. At that time the lesions were sharply marginated and a few of them were on an erythematous base. The lesions were noted to follow the embryonic lines of cleavage. In order to rule out a fungus infection, a culture was taken on November 20, 1959, using Sabouraud's Dextrose Agar. She was given some general quartz light and Pragmatar was prescribed to be applied to the lesions at night. The culture read on November 25, 1959, was negative for fungi.

When seen on November 27, 1959, the eruption was essentially unchanged. At this time the Pragmatar was discontinued and Aquaphor and cold cream in equal parts prescribed.

On December 4, 1959, the child had developed new lesions on the right and left chest as well as on the thighs anteriorly. Also they were present on the face, chest, abdomen, back, and anterior thighs.

On December 9, 1959, the child was taken to a Well Baby Clinic. Dr. Ernest L. Williams, Jr., Chief of the Dermatologic Service, Kern County General Hospital, in charge of the Well Baby Clinic, made a diagnosis of pityriasis rosea.

Although I felt sure this was pityriasis rosea, I sent the child to Dr. Henry G. Morgan, a Los Angeles dermatologist, on December 12, 1959. He also made a primary diagnosis of pityriasis rosea.

Examination on December 12, 1959, revealed hypopigmented, maculo-squamous lesions with fine, flat, superficial scales in a state of involution involving the chest, trunk, face, and upper parts of the extremities. There were no ring scales adherent to the peripheries. Cortisone cream for topical application to the involved areas and colloidal oatmeal preparation for baths were recommended.

On December 18, 1959, cultures were taken from 3 lesions and again Sabouraud's Dextrose Agar was used. Cultures read on December 24, 1959, were again negative for fungal infection. On December 18, 1959, although there was no family history of syphilis and although there was no clinical evidence of syphilis a serology was done since the secondary eruption of syphilis may be confused with the secondary eruption of pityriasis rosea. Both the VDRL and Mazzini tests were non-reactive. Complete blood count at this time was an follows: Hemoglobin concentration 9.1 Gm./100 c.c. (57.7%); white blood count, 5,800 per cubic millimeter; differential count, polymorphonuclear leucocytes, 39, stabs, 3, lymphocytes, 55, and monocytes 3; Hematocrit, 32%.

On January 7, 1960, the lesions were beginning to fade out but the areas of depigmentation could still be clearly seen.

When seen on February 8, 1960, the lesions had almost completely disappeared. At this time she weighed 16 pounds and 8 ounces which was very good considering her birth weight of 4 pounds and 8 ounces.

The child had a mild upper respiratory infection on February 18, 1960. When seen on this date it was almost impossible to detect the areas involved by pityriasis rosea. At this time the child was given Elixir of Feosol for iron deficiency anemia. She was last seen on May 3, 1960, and at this time was in excellent condition.

DISCUSSION

Age incidence. According to Crissey pityriasis rosea is largely a disease of young adults and children. From 1008 unselected cases he reported that 76 per cent of cases occurred before the age of 35 and 26 per cent before the age of 20 years. Other investigators have reported age incidence of the disease. The following table summarizes the youngest patients reported by various investigators.

TABLE 1

Investigators	Age of youngest patients
Crocker	7 months
Weiss, Lane and Showman	8 months
Savill	11 months
Crissey	1 year
Fox	17 months
Little	18 months
Leiner	21 months
Thomson and Cumings	21 months
Adamson	2 years
Lord	3 years
High and Rulison	4 years
Niles and Klumpp	5 years

The oldest patients reported with pityriasis rosea were a 70 year old patient of Crocker and a 77 year old of Thomson and Cumings.

Sex. Weiss, Lane, and Showman, Towle, Thieberge, Moingeard, Adamson, and Crissey report ratios of 2 females to one male with pityriasis rosea. Fifty seven and seven-tenths of the cases of Highman and Rulison were females and 42.3 per cent were males. It is important to note that Little, Crocker, and Cunningham found that their cases occurred with equal frequency in the two sexes. It is the consensus of opinion that the disease is twice as frequent in females as in males.

Race. In 1940 Niles and Klumpp stated that Pityriasis rosea was usually seen in white persons. Fox in 1908 reported on 2200 cases of cutaneous diseases in white persons and the same number in Negroes. He found pityriasis rosea in 7 white patients and 6 Negro patients. Cassey states that no demonstrable racial predisposition exists. Whites, Negroes, Orientals, and American Indians are affected.

Differential Diagnosis. Differential diagnosis include: (1) Psoriasis. (2) Secondary eruption of syphilis. (3) Ringworm. (4) Lichen planus. (5) Seborrheic dermatitis and (6) Scabies.

In this case *psoriasis* could be excluded for several reasons. In the first place the typical imbricated mother-of-pearl type scale seen in psoriasis was not present. The scale in this child was salmon colored. In the second place the Auspitz phenomenon (tiny bleeding points seen when the scale is removed) was absent. In the third place the lesions of psoriasis commonly appear in the scalp and typical plaques can often be seen on elbows, knees, and in sacral areas.

In *secondary syphilis* the lesions frequently appear on the palms and soles. The lesions of pityriasis rosea do not usually appear in these areas. In this infant the plaques followed the lines of cleavage. The secondary eruption of syphilis does not regularly follow the lines of cleavage. The VDRL and Mazzini tests were negative.

Ringworm lesions bear no relation to the lines of cleavage whereas has been forestated the lesions in this child followed the lines of cleavage. There are rarely more than 5 or 6 lesions of ringworm upon the body. This child had numerous lesions. In general the lesions of ringworm are on the exposed parts of the body whereas those of pityriasis rosea are usually upon the covered parts of the body. This infant had lesions on the trunk and extremities. The

lesions of pityriasis rosea do not usually involve the face but the eruptions appeared on this infant's face. Cultures disclosed no fungi.

Lichen planus may be confused with the papular type of pityriasis. In this patient the papular ash was not present and consequently there was no differential problem regarding lichen planus.

Seborrheic dermatitis develops more slowly than does pityriasis rosea. In seborrheic dermatitis there are usually greasy, scaly lesions found in the scalp, eyebrows, behind the ears, the nasolabial folds, the sternum, spinal column, the umbilicus, between the scapulas, the flexures of the extremities and creases of the groin. The lesions of seborrheic dermatitis do not follow the lines of cleavage. The scales of pityriasis rosea are not as coarse as those of seborrheic dermatitis. There was nothing in this infant to indicate seborrheic dermatitis.

In *scabies* there are sites of predilection that help differentiate this disease from the papular type of pityriasis. Burrows are often seen in scabies and there is a tendency for the itching of scabies to become worse at night. Since this child did not have the papular type of pityriasis rosea, scabies did not enter in the differential diagnosis.

SUMMARY

1. A 3 month old female with pityriasis rosea is presented. It is felt that this is the youngest person ever reported with this disease.
2. Pityriasis rosea seems to have no racial predisposition.
3. Pityriasis seems to be a disease of young adulthood and children for the most part but it has been reported in a patient 77 years old.
4. According to some investigators pityriasis rosea is more common than it was 40 years ago.
5. Most investigators have found the ratio of 2 females to 1 male in pityriasis rosea.
6. Differential diagnosis of pityriasis rosea include: psoriasis, secondary eruption of syphilis, ringworm, lichen planus, seborrheic dermatitis, and scabies.

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Pediatric Conference . . .

THE DRISCOLL FOUNDATION
CHILDREN'S HOSPITAL
CORPUS CHRISTI, TEXAS

CHILDHOOD TUBERCULOSIS
January 26, 1960

J. M. SLOAN, M.D., *Medical Director, Presiding*
RUSSELL BLATTNER, M.D., *Professor and Chairman of the Department of Pediatrics*
KATHERINE H. K. HSU, M.D., *Assistant Professor of Pediatrics; Baylor University College of Medicine, Houston, Texas, Guests*

DR. SLOAN: We are fortunate in having Dr. Blattner and Dr. Hsu with us for this conference. Dr. Blattner will discuss the broad aspects of the problem of childhood tuberculosis, and Dr. Hsu who is well known for her work as Director of the Children's Division of the Section on Tuberculosis at Jefferson Davis Hospital, Houston, Texas will discuss the management of childhood tuberculosis.

Most of the children of indigent parents in a five county area are hospitalized in this hospital. It is unusual for an acute children's hospital to care for so many children with tuberculosis.

The following slides represent our experience in patients discharged from 1953 to January 1, 1959.

TABLE 1
Ratio of Tuberculosis Patients to All Patients

	Discharged No.	*%*
All patients	5,237	100.0
T. B. patients	199	3.7

TABLE 2
Ratio of Patient-Days

	Discharged No.	*%*
All patients	170,479	100.0
T. B. patients	48,328	28.3

Thus, although admissions for T.B. were less than 4%, these represent over one fourth of the total patients days, and thus roughly one fourth of the operating expense of the hospital.

TABLE 3
Distribution of Race

	No.	*%*
Latin-American	187	94.0
Anglo-American	9	4.5
Negro	3	1.5
	199	100.0

TABLE 4
Distribution of Age

Year	*No. of Cases*	*Year*	*No. of Cases*
1st	32	9th	10
2nd	38	10th	3
3rd	26	11th	2
4th	18	12th	3
5th	15	13th	4
6th	15	14th	8
7th	11	15th	2
8th	9	16th	5
			199

Remark: Indicating the age at the time of admission

This shows quite clearly that childhood T.B. is often a disease of infancy. Certainly a great many and probably a majority of the children admitted in the 2nd-3rd-4th year developed their primary infection during the first year.

TABLE 5
T. B. Contact

Contact unknown		64 cases
Contact known		135 cases
Parents	79	
Grandparents	26	
Siblings	8	
Others	37	

Remark: 18 *cases had more than one known contact.*

TABLE 6

	No.	*%*
Patients with one form of lesion (e.g. Pulmonary)	162	81.5
Patients with multiple forms of lesions (e.g. Pulmonary plus meningitis)	37	18.5

TABLE 7
Distribution of Lesions

Pulmonary			167
Non-Pulmonary			68
T. B. Meningitis		25	
Skeletal System		16	
Vertebrae	12		
Hip	1		
Pubis	1		
Femur	1		
Knee	1		
Lymphatic System		17	
Cervical lmyph node	14		
Preauricular lymph node	2		
Inguinal lymph node	1		
Eye		5	
Ear, middle		2	
Larynx		1	
Tonsil		1	
Peritoneum		1	
		68	
Generalized Dissemination			2
(Types of lesions in 199 cases)			237

The resident staff will present three cases.

CASE PRESENTATION I
H-3527

DR. RENE RODRIGUEZ, 1st year pediatric resident: This 6-year-old Latin-American female was admitted on 12-4-57 because of swelling of the abdomen, legs and hands.

Present History: Illness began 3 months prior to admission with poor appetite, loss of weight, a colicky type of abdominal pain, and diarrhea with 7 or 8 green, liquid stools daily without nausea or vomiting. Swelling of the abdomen occurred about 1 month prior to admission followed by swelling of the legs and hands. The edema was progressive, as well as the pallor and cachexia. There was no history of fever. She has had oliguria with smoky urine, and dyspnea since the onset of the edema.

Past History and Developmental History: Not contributory.

Family History: Mother has advanced pulmonary T.B. 2 siblings have primary pulmonary T.B.

Case I H-3527, Fig. 1. On Admission 12-5-57, Severe malnutrition with Nutritional Edema. Serum Proteins 4.7 AIB 1.7 Globulin 3.0 Wt. 39 lbs.
Case I, Fig. 2. 12-18-57 Wt. 26 lbs.

Physical Examination: Weight 39 lbs., height 40¼ inches, temperature 102°, pulse 125, respirations 36, and blood pressure 100/65. The child was acutely and chronically ill on admission and was irritable and apprehensive. She had enlargement of the abdomen with ascites, and edema of the lower extremities. There was

slight edema of the face and hands. Some petechiae were present on the abdomen and back. There were prominent veins over the lower chest and upper abdomen. There were many small discrete cervical nodes. Decreased breath sounds and dullness in both bases, more marked on the right.

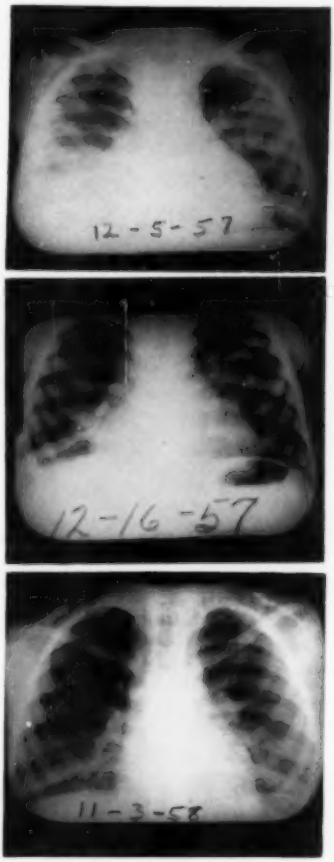
Laboratory Findings: The RBC was 2,370,000, the WBC 24,250 with 54 segs, 5 stabs, and 40 lymphocytes. The hemoglobin 46%, the hematocrit 22%. Total serum proteins: on admission 4.7 gm.%, albumen 1.7 gm.%, globulin 3 gm.%, AG ratio 0.57, cholesterol 46 mg.%. Cephalin flocculation and transaminase were within normal limits. Thymol turbidity 6.8 units, sedimentation rate 31 (micro), CRP 4+, O.T. 1:1000 positive (15 mm.). Gastric washings positive for T.B. by culture. Paracentesis: peritoneal fluid reported negative for acid fast bacilli in smear and culture, positive in Guinea pig inoculation. Thoracentesis fluid was sterile in culture, and smears were negative for acid fast bacilli.

Case No. I. Fig. 3.
10-29-58 Wt. 48 lbs.

Hospital Course: The patient was started on anti-tuberculous drugs on 12-6-57, two days after admission; with INH, 5 mg./kg./day, increased progressively to 25 mg./kg./day during the first month and continued at this dose for 1 month; then decreased to 18 mgs./kg./day for 5 months and then 12 mgs./kg./day for a total of 18 months. Streptomycin, 30 mg./kg./day was given daily for 3 months; 3 times a week for 3 months and then 2 times a week for 2 months and was discontinued on 8-28-58 after 8 months of treatment. PAS, 200 mgs./kg./day was given for 18 months. The child showed progressive improvement and was discharged July 2, 1959 after 18 months hospitalization, weighing 56 lbs. 12 oz. (Case I, Fig. 1, 2, 3.)

DR. SLOAN: Dr. H. R. Arnold, our chief of radiology will present the films on this case.

DR. ARNOLD: The initial film of the abdomen was made on admission. The abdomen shows diffuse haziness throughout and we can see bulging of the flanks on both sides. This is the picture one sees with ascites or a peritonitis. In addition, we see multiple gas-filled loops of large and small bowel and dilatation of the stomach suggesting adynamic ileus. Here is the initial chest film which was made on 12-5-57. We see bilateral pulmonary infiltrations and in addition we see a rather large obstructive pneumatocele in the apical portion of the left lung field. In the right pleural space there is a moderate amount of effusion present. There is also bilateral hilar adenopathy. A decubitus film was made to demonstrate the extent of this pleural effusion and to see if we were dealing with a free effusion or simply thickened pleura. Here we see the large obstructive pneumatocele. This next film on 12-10-57 shows a progression of the pleural effusion and the pneumatocele shows an increase in tension. There are other smaller pneumatoceles developing in the left lung field. On this film, on 12-16-57, we see a marked decrease in the amount of effusion on the right. There is still evidence of an infiltration and some accompanying atelectasis in the right lower lung field and the pneumatoceles are still present in the left lung field along with the infiltration. This is simply a progress film again showing much of the same thing we saw on the previous film. The pneumatoceles are still present although they have decreased in tension. Here is a film on 12-20-57.



Case I, Fig. 4. 12-5-57
Case I, Fig. 5. 12-16-57
Case I, Fig. 6. 11-3-58

Now, we can see that the pnuematoceles have subsided and are no longer evident. There is still a diffuse infiltration in the left lung field and also an infiltration in the right lung field with atelectasis and some accompanying pleural reaction. On 2-18-58 we begin to see improvement in the chest. There is a decrease in pleural reaction and the infiltrations seem to be subsiding bilaterally. Now we have a series of films which simply show progress improvement. Here is a film on 3-5-58, another on 6-27-58, showing progressive clearing of the bilateral infiltrations. On 11-3-58 we see further resolution of the infiltrations and the pleural reaction. They have completely subsided and the hilar adenopathy is also subsiding and on 12-14-59 we thought that the lesions in the lungs had reached stabilization and there was no longer evidence of any activity. (Case I, Fig. 4, 5, 6).

DR. SLOAN: This case illustrates a severe disseminated form of the disease without miliary or central nervous system or bone involvement. It also illustrates a good response to specific therapy. If we treated this child today, we would have used larger doses of INH (to 40 mg./kg. initially) and smaller doses of Streptomycin (20 mg./kg.) over a shorter period of time (about 2 months).

CASE PRESENTATION II

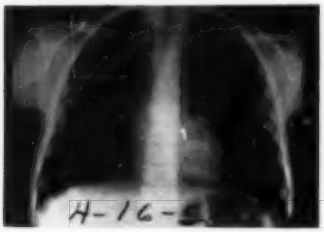
H-220

DR. EDUARDO MUNOZ, 3rd year pediatric resident:

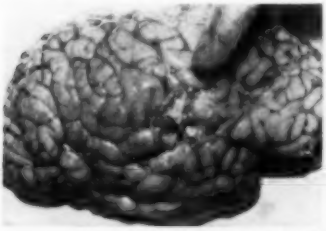
This patient was admitted to the hospital for the first time in June of 1953 at the age of 2 years with the diagnosis of pulmonary tuberculosis. This patient's mother had active pulmonary tuberculosis. He remained in the hospital for 18 months; during this time his treatment consisted of rest and good general care. No specific tuberculous antimicrobial drugs were given. On discharge he was markedly improved and had gained weight from 21½ to 31 lbs. Improvement in the x-ray appearance of the chest was very slow, but on discharge x-ray showed fibrosis and no active disease. He was followed in the T.B. Clinic regularly from December 16, 1954 to Septmber 27, 1957. During this time, 5 x-rays were reported "stabilized process"; the last on September 27, 1957. His next admission was on April 22, 1958 at the age of 8 years. At that time, he had tuberculous meningitis. X-ray of the chest was negative. The meningitis was severe with coma and severe spastic contracture of the extremities. He was treated with three anti-tuberculous drugs, PAS (200 mg./kg. of body weight) daily and INH (20 mg./kg. of body weight) daily for a period of 15 months. Strepto-

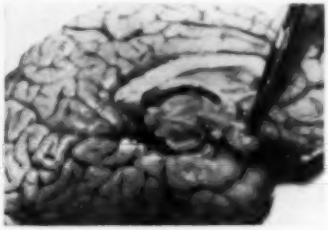
mycin, 1 gm. daily was administered for 3 months; then 1 gm. twice a week for 10 months; then 1 gm. daily for two months. In addition he received Hydrocortisone (Solu-cortef) intrathecally, 20 mg. daily for 9 days from 6-17-58 to 6-25-58. He slowly improved and after 5 months, was partially ambulatory with severe motor residual, mental impairment and frequent convulsions. He relapsed after 15 months of treatment and died July 17, 1959. Autopsy disclosed (1) tuberculous meningitis, (2) bilateral bronchopneumonia, (3) pulmonary hilar lymphadenopathy, and (4) multiple fibrocaseous lesions of the brain.

Case II, H-220, Fig. 1. 7-17-53. Diag: Pulmonary T.B.
Case II, Fig. 2. 12-16-54. Showing slow progressive improvement, "Stabilized". Has had no specific "anti T.B." drugs.



Case II, Fig. 3. 4-16-58. On Admission with T.B. Meningitis. "No X-ray evidence of active disease".
Case II, Fig. 4. 6-10-59. Chest Film one month before death due to T.B. Meningitis. Still shows no evidence of active T.B.





Case II, Fig. 5. Tuberculoma of Cortex.
Case II, Fig. 6. Tuberculoma of Optic Chiasm. Note: Normal Ventricular System.

DR. ARNOLD: On one of the initial films made of the chest we see bilateral hilar adenopathy and infiltration which is compatible with an active primary complex. The following films will show a very slow but progressive improvement in the chest. Here on 10-19-53; again we see evidence of bilateral disease without much change over the past two month period. On 12-30-53, again we see evidence of bilateral disease without much improvement. Over the whole series of films we see a slow progressive change. Here on 5-3-54 and finally on 10-1-54 we felt that the pulmonary lesions had reached stabilization and there was no longer any evidence of activity. On 12-16-54 this was the appearance of the chest when the patient was discharged from the hospital. Here is the appearance of the chest when the patient was readmitted with tuberculous meningitis. Again, there is no evidence of any active pulmonary disease. This is the last film on 6-10-59, again showing no evidence of active pulmonary disease and this was made shortly before the death of this child. (Case II, Fig. 1, 2, 3, 4, 5, 6.)

DR. SLOAN: This case represents a fatal complication which almost certainly could have been prevented had our present concept of management been applied in 1953. It also represents a case of T.B. meningitis which was resistant to treatment. The cause of death was also unusual, in that the child had persistent parenchymal lesions in the brain, but did not have the internal hydrocephalus which is so common in fatal cases of T. B. meningitis.

CASE PRESENTATION III
H-4898

DR. LORENZO RIVERA, 2nd year pediatric resident:

This patient was admitted on June 23, 1959 with the chief complaint of limping and pain in the left hip since May of 1959.

Present Illness: This is a 5-year-old Latin American female who fell in the bathtub during the latter part of May, 1959 injuring her left hip. Following this injury, she had pain and slight swelling of left hip and walked with a limp. She was seen in the clinic on June 17th because of pain and limping of the left leg.

Family History: Not contributory. No history of T.B. contact. Three siblings—all had tuberculin patch tests which were negative.

Past History: Not contributory.

Physical Examination: Weight 31 lbs., height 37½ inches. There was swelling, slight pain and tenderness over the left hip which was

held in slight external rotation, and there was limitation of motion in external rotation. The patient walked with a limp.

Laboratory Findings: Hemogram was normal, CRP 1+, sedimentation rate 28, T.B. patch test was positive (30 mm.), O.T. 1:10,000 positive (30 mm.), Coccidiodin, histoplasmin, toxoplasmin skin tests were negative. Cultures of gastric washings and urines were negative for acid fast bacilli. X-ray—Chest negative. Hip—tuberculosis involvement of the left hip.

Hospital Course: The hip was immobilized and anti-tuberculous drugs, INH, PAS, and streptomycin were administered. The dosages:

INH 20 mg./kg. (100 mg. t.i.d.) until 11-13-59.
PAS 200 mg./kg. body weight (0.5 gm. t.i.d.) until 11-13-59.
Streptomycin 30 mg./kg. body weight (0.5 gm.) daily to 10-13-59.
Streptomycin 30 mg./kg. body weight, t.i.w. from 10-13 to 11-13-59.

On 11-7-59 a biopsy of the left hip was performed and microscopic examination revealed a chondroblastoma.

Case III, H-4898, Fig. 1. Diag. T.B. Hip. 6-17-59.
Case III, Fig. 2. 8-24-59. Progression of lesion despite Treatment for T.B.

Dr. Arnold: The initial film of the left hip was taken on 6-17-59—On this film we see a destructive lesion which involves both the metaphysis and epiphysis of the left hip and there is a small central nidus suggesting a sequestration. At this time this lesion had all the characteristics of an inflammatory lesion and on the basis of the positive tuberculin test, we felt that this was a tuberculous lesion in origin. On 8-24-59 we see that this lesion shows progression. The lesion had extended laterally and superiorly and we see some sclerosis developing. On 10-20-59 we see further progression of this lesion and now the lesion is developing well defined margins surrounded by a sclerotic zone. In view of this progression while on tuberculous therapy and in view of the appearance the lesion now has, a chondroblastoma was suspected rather than the original diag-

nosis of a tuberculous lesion. It was at this time that a biopsy was recommended for confirmation of the radiographic diagnosis. This is the appearance of the lesion after biopsy and curettage; essentially the same but perhaps the margins are somewhat sharper than they were on the previous film one month ago. (Case III, Fig. 1, 2, 3, 4.)

Case III, Fig. 3. 10-20-59. Chondroblastoma Suspected.
Case III, Fig. 4. 12-23-59. Biopsy 11-7-59. Diagnosis: Chondroblastoma.

DR. SLOAN: A positive tuberculin, plus disability in a hip which on x-ray showed pathology compatible with the diagnosis of tuberculosis of the hip led to intensive therapy for T.B. X-ray evidence of progression of the hip pathology and a change suggestive of tumor rather than T.B. led to biopsy and to the correct diagnosis. However, even though the diagnosis of T.B. of the hip has been ruled out, because of the strongly positive tuberculin test, INH, 10 mg./kg. body weight is now being given and it is planned to continue this therapy until June 1960.

DR. BLATTNER: The field of tuberculosis is one in which many important advances have been made; however much remains to be done, not only in gaining new knowledge, but in the effective application of already known facts. The basic understanding of pathogenesis, significance of the proliferative and exudative manifestations of the disease process, and a clarification of the relationship between immunity and hypersensitivity provide us with a firm foundation and broad understanding with which to approach the many and often complicated problems of infection caused by the acid fast mycobacteria.

As the years have passed we have been adding to this fund of knowledge in a number of significant ways, and I should like to refer to several of these. An important advance, I think, is in the

field of microbiology. We have learned a great deal about the culture of the acid fast micro-organisms and on the basis of this knowledge, we have been able to develop a convenient and useful classification of the acid fast organisms. We have learned that these micro-organisms vary as to antigenicity, cultural requirements and in their capacity to produce disease. It is increasingly clear that the several members of this broad group produce different pathologic responses depending not only on the host but on the nature of the microorganism itself. The importance of the chromogenic acid fast organisms has been emphasized in recent literature.

In the field of tuberculosis control it is perhaps of some value to recall certain experiences of the past. I am sure that many of us can remember the so-called "galloping consumption" in the young adult, and we also recall the era when tuberculous meningitis was generally regarded as an incurable disease. A few years ago these unpleasant features of tuberculosis were taken for granted. A favorite cliche was "it just runs in the family." I remember a girl in our neighborhood who had had tuberculosis of the hip. She was always in fair health, but eventually died in her teens of "galloping consumption." As I remember the incident, everyone thought that her fate was inevitable and that the only explanation for her demise was that consumption ran in her family! As a child I also remember vaguely that there were institutions to which people with tuberculosis were sent. In most instances we never saw them again since most of them seem to have died. Now-a-days our approach to tuberculosis is much more optimistic and the tools for eventual control are powerful indeed.

Elimination of tuberculosis in a well supervised community by means of excellent public health measures has been demonstrated over and over again. An excellent example of this is the fine tuberculosis control program carried out in Minnesota by Dr. J. Arthur Myers, the health authority in that State. He has been able to eliminate bovine tuberculosis, and has succeeded in reducing human tuberculosis quite effectively, using improved methods for case finding, isolation of open cases, medical treatment, surgical treatment of open cases, etc. Tuberculosis incidence in that State has been reduced almost to zero. Similar success is reported in such enlightened countries as Holland and Denmark. In general, these advances were made on the basis of public health measures: improved sanitation, better nutrition, adequate housing, improvement of dairy herds, etc.

Another aspect of control involves the use of active immunization against tuberculosis utilizing the vaccine "B.C.G.", Bacillus of Calmette Guerin. This mode of control has never been popular in the United States. However, it has been used extensively in South America and in other countries of the world with considerable success. At the present time, safe and effective B.C.G. vaccine is available, and it most certainly has a place in the world-wide control of tuberculosis, its use depending to a large extent on local and environmental conditions. Active immunization with B.C.G. is quite effective in converting a negative tuberculin test to a positive one, and in producing a certain degree of immunity. In England, Wilfrid Gaisford, demonstrated that B.C.G. inoculation of newborn babies resulted in the development of a positive tuberculin test within a period of four weeks, and that their resistance to infection was increased even when the infants were exposed to an environment where the acid fast organism could not be eliminated. His figures indicated that B.C.G. vaccination during infancy did result in a decreased incidence of tuberculosis in the vaccinated group. However, there are disadvantages in the use of B.C.G. I am sure that Dr. Hsu will refer to this problem in her discussion.

Recently, we have come to realize that the tuberculin test properly used is an extremely important and sensitive method for the detection of tuberculous infection.

If we consider tuberculosis as a world-wide problem our approach to treatment and control will be determined to a large extent by the general conditions which exist. For example, where a high percentage of the population is infected with tuberculosis, and where malnutrition, poor housing and poor sanitation conditions are common, one must approach the tuberculosis problem quite differently from the approach used in Sweden or in Minneapolis, Minnesota. On a recent trip to Canada, Dr. Sloan and I were interested in visiting a hospital in Montreal, The BCG Clinic of Montreal, Inc., where newborn babies, born to mothers whose home environment would have permitted unavoidable exposure to tuberculosis, are admitted and boarded. The infants admitted to this special hospital are inoculated with B.C.G., and are not permitted to return to their homes until each infant has a positive tuberculin test. Every effort is made to provide proper care for the individuals who are the source of infection in the home, but in some instances, according to our informants, it was not possible to remove all of these people from all the homes. It was the opinion of workers in this institution that

such measures reduce the incidence of tuberculosis in this particular population group. It was interesting that the need for this hospital is gradually lessening each year as adequate tuberculosis control in the community is developed.

When our group in Houston, Texas, first started to work on the problem of tuberculosis, the situation was a bad one. The available facilities were grossly inadequate and case finding was inefficient. It was disheartening indeed to observe families with tuberculosis go without adequate supervision or care and to see the infants and young children developing active tuberculosis. Through the efforts of a number of people, the officials in our community became aware of the deplorable conditions and determined to do something about it. An important step was taken when it was agreed that a full-time pediatrician be hired who could devote all efforts toward solving some of the problems of tuberculosis in our city. Dr. Katherine H. K. Hsu came to our city with an excellent background as a specialist in tuberculosis. She joined forces with us, and since her arrival much has been accomplished. We learned that the incidence of tuberculosis in our community was much higher than previous reporting indicated. Now, with the development and availability of effective antimicrobial agents, it is my opinion that we can anticipate even greater progress in years to come. It is with great pleasure, Dr. Sloan, that I introduce Dr. Katherine H. K. Hsu, Assistant Professor of Pediatrics, Baylor University College of Medicine, and Director of the Harris County Pediatric Tuberculosis Clinic. She is widely regarded as an expert in pediatric tuberculosis and has received recognition not only in this country but in many other parts of the world as well.

Dr. Hsu: I appreciate very much the opportunity of participating in this panel for the discussion of the problems of tuberculosis in children. This is a very timely subject, because in recent years there has been a drastic change in the management of tuberculosis in children. The advent of potent anti-tuberculosis drugs has made possible, for the first time in history, a direct attack on tubercle bacilli. Among the drugs discovered, Isoniazid (INH) is of special interest to the pediatricians. It is a very potent anti-tuberculosis drug which can be taken by mouth. Mothers can give the drug to the children without trouble because there is no unpleasant odor or taste. It is inexpensive; therefore, it is available even to the poor.

The discovery of INH has marked a great advance in tuberculosis control in children. It has proved to be the most effective therapy

for manifest forms of tuberculosis. Of greater significance, is its effectiveness in preventing the development of serious manifestations of tuberculosis in infected children. All the dreadful consequences of primary tuberculosis such as meningitis, miliary tuberculosis, bone and joint tuberculosis, and progressive endothoracic tuberculosis, can be prevented by early institution of INH therapy. The efficacy of this preventive effect of INH has been well demonstrated by the recent nation-wide controlled study by the USPHS, and likewise by the large-scale study made by the National Institute of Hygiene of France. In Houston and Harris County, Texas, a study of the early INH therapy of primary tuberculosis was started in 1953 at the Houston Children's Tuberculosis Clinic. The result has been most gratifying. After 7 years of continuous study, there is no doubt in my mind of the great preventive value of INH. Therefore, I would recommend the application of this method of tuberculosis control in children to the widest possible extent.

A number of questions have frequently been asked regarding the management of tuberculosis in children. I shall attempt to answer a few of them this evening.

I. *What is the Best Way of Detecting Tuberculosis in Children?*

X-ray examination has only very limited value for detection of primary tuberculosis in children because the great majority of cases do not show any x-ray manifestations.

The tuberculin test reveals primary tuberculosis in practically all cases. A positive reaction indicates the presence of living tubercle bacilli in the child's body, therefore pointing to the need for further examination.

II. *What is a Positive Tuberculin Reaction?*

The intradermal test is the standard technique for tuberculin testing. The purified protein derivative (PPD) at the dosage of 0.0001 mg. is widely used the world over, although certain clinics prefer the use of the Old Tuberculin (O.T. 1:1000). The test is read 48 to 72 hours later. Induration of 6 mm. or more is a positive test. A large and intensive reaction indicates greater liability of progressive disease than a small reaction.

Vollmer's patch test is convenient to use, but in our experience it missed about 50% of positive Mantoux reactors. The recently developed multiple-puncture Heaf test is a far superior test for screening purposes.

III. *What are the Indications for Anti-Tuberculosis Therapy in Children?*

Drug therapy is recommended in the following types of cases:

1. Children less than 36 months of age with a positive Mantoux test to 0.0001 mg. PPD.
2. Children older than 36 months of age whose tuberculin reactions have converted from negative to positive within a year.
3. Positive tuberculin reactors with a positive culture of of tubercle bacilli from gastric contents, urine or biopsy specimens.
4. Positive tuberculin reactors showing x-ray indication or clinical manifestations of active tuberculosis.

Latent tuberculous infection is the precursor of manifested tuberculous disease. Children with an active infection should be given the benefit of anti-tuberculosis drug therapy whether it is clinically latent or manifested.

Corticosteroids are known to reactivate old tuberculous lesions. All children who, for any reason, need a prolonged course of corticosteroid therapy should be tuberculin tested first. If the reaction is positive, INH therapy should be given, even though the tuberculosis is considered inactive. The duration of INH therapy should extend at least 3 months beyond the corticoid therapy.

IV. *What is the Recommended Drug Therapy for Tuberculosis in Children?*

Isoniazid (INH) is the most potent and the least toxic of anti-tuberculosis drugs. *It is essential to include INH in any treatment regimen of active tuberculosis.* The duration of therapy should be at least one year. The administration of pyridoxine for the prevention of neuritis does not appear to be necessary in children.

1. *For Active Primary Infection without X-ray or Clinical Manifestations,* such as in young children under 36 months of age or older children who have recently converted to positive tuberculin reaction, INH alone is considered satisfactory treatment.

Dosage: INH 15-20 mg/kg/day in 2 doses
PAS Not essential.

2. *For Localized Diseases* such as lymphadenitis and segmental disease of the lung, use INH combined with PAS.

Dosage: INH 15-20 mg/kg/day in 2 doses
PAS 200 mg/kg/day in 2-3 doses
(Maximum 12 gm)

3. *For Miliary Tuberculosis* use triple drug therapy.

Dosage: SM 20 mg/kg/day
(Maximum dose 1.0 gm/day)
INH 30-40 mg/kg/day until miliary densities clear up on the radiograph. Reduce dosage to 20 mg/kg thereafter.
PAS 200 mg/kg/day
(Maximum dose 12 gm/day)

Duration:

SM not longer than one month, if there is satisfactory improvement.

INH and PAS for at least one year from the time the miliary densities clear up in the chest radiograph.

4. *For Tuberculous Meningitis* use triple drug therapy.

Dosage: SM 20 mg/kg/day
(Maximum dose 1.0 gm/day)
INH 30-40 mg/kg daily until spinal fluid becomes normal, then reduce dosage to 20/mg/kg. (In children with vomiting, parenteral administration of INH is advised to assure adequate intake).
PAS 200 mg/kg/day
(Maximum dose 12 gm/day)

Duration:

Streptomycin to be discontinued within one month after good clinical improvement.
Continue INH and PAS for at least one year from the time the spinal fluid returns to normal.

Corticosteroid as Adjuvant to Antimicrobial Therapy.

Corticosteroid may hasten the resolution of the following types of disease condition:

Pleurisy with massive effusion.
Obstructive emphysema or atelectasis due to large hilar nodes or endobronchial tuberculosis.
Miliary tuberculosis.
Tuberculosis meningitis.

Dosage:

Prednisone 1 mg/kg/day for 4-6 weeks.
For tuberculous meningitis, hydrocortisone (Solu-cortef) 2.5—5 mg/day given intrathecally for 2-4 weeks.

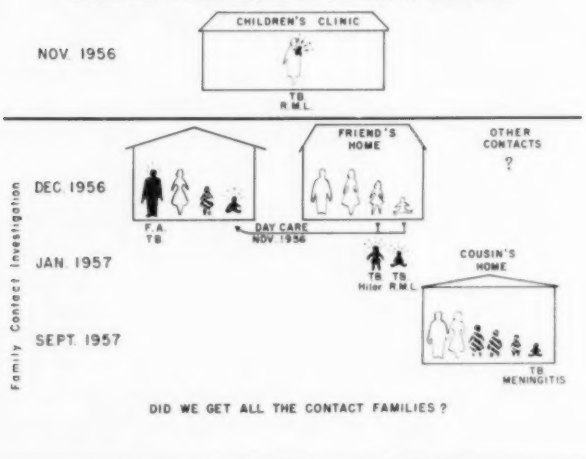
PRESENT DAY SITUATION OF TUBERCULOSIS IN CHILDREN IN THE U. S.

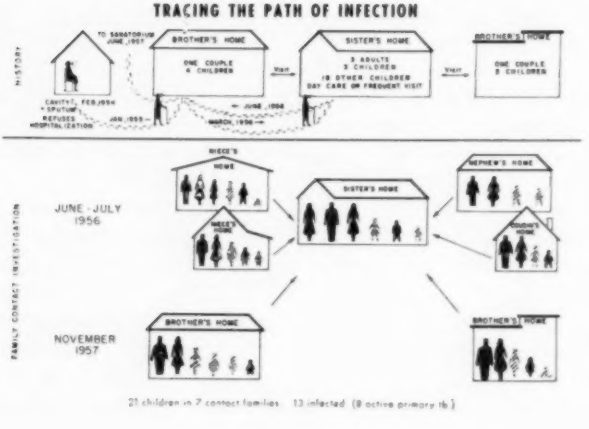
In the past 50 years there has been a dramatic decline in the prevalence of tuberculosis in this country. This change is well reflected in the tuberculous infection rate in children. For instance, Dr. Sloan told me this afternoon that in the early thirties there was a tuberculin survey of school children in Corpus Christi, in which the majority of children were positive reactors. Is that right?

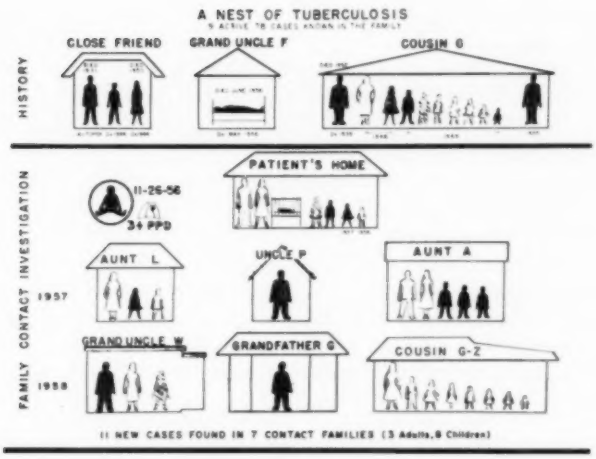
DR. SLOAN: Up to 87% in high school children using the Mantoux test with 1:1000 O.T.

DR. HSU: Today there are only 5% to 8% of positive reactors in white Navy recruits, according to a study by the USPHS. Recent tuberculin surveys conducted in the public grade schools in Houston and Harris County yielded only 2% to 6% positive reactors in the poor resident districts. This low infection rate in children and young adults is an excellent indication of the success of tuberculosis control in the general population. However, it is important to know that although the flood of tuberculosis is over, there remain many small pools of tuberculosis in the community. Within these pools, tuberculosis is still very prevalent; and as long as they remain, they will continue to breed tuberculosis and perpetuate the dissemination of the disease in the community. To illustrate the seriousness of the problem of "tuberculosis pools" I will show the following three slides. All of these cases were taken from the contact investigation study at the Houston Children's Tuberculosis Clinic.

Note: Solid gray — No tuberculosis.
Solid Black — Active tuberculosis.
Black striped — Positive tuberculin reaction.
Dotted radiating lines — Positive sputum or gastric.







This knowledge of the focal distribution of tuberculosis is of utmost importance for the eradication of tuberculosis. It stresses the importance of examination of contacts whenever a new case of tuberculosis is discovered. In our experience, the infection rate is very high among children who have been in contact with adults newly found to have tuberculosis. Early INH therapy in these recently infected children will save them from destructive tuberculous diseases.

SUMMARY

This discussion has been of the advances in the detection and management of tuberculosis in children in very recent years. Many current studies now under way will give us more to learn in the near future.

DR. SLOAN: The conference is now open for questions.

QUERY: I want to ask a question which will probably be very difficult to answer. How do you know when to stop treating T.B. meningitis?

DR. HSU: This is a question which is hard to answer because the extent of the disease in the brain cannot always be judged from the spinal fluid examination. However, for clinicians, spinal fluid findings and focal neurologic signs are the best guides for treatment. We have been treating tuberculous meningitis for at least

one year after the spinal fluid has returned to normal. No relapse has been noticed so far.

QUERY: Thank you. Now, may I ask another question while I am here? Now, about the dosage in primary tuberculosis. You mentioned 20 mgs./kilo body weight of INH?

DR. HSU: Yes, actually the optimum dosage of INH depends upon the individual child. The reason for a higher dosage is because certain children inactivate the daily dose of Isoniazid in the body. Since we do not know which children are quick inactivators, it is safer to use a higher dose than to use a lower dose. Fortunately, Isoniazid is such a non-toxic drug that even with a dosage of 20 mgs./kilo per day, we have not seen any toxic effect. Drug idiosyncracy was seen in only 3 or 4 children who developed skin rash.

QUERY: Would you say a word about the tuberculin testing materials—patch test, PPD and old tuberculin?

DR. HSU: For intradermal tests, one may use either PPD or OT. The only drawback of the OT is the lack of uniformity of potency from batch to batch. The potency of PPD is standardized; therefore, it has been recommended by the World Health Organization as the international standard. For tuberculin surveys, 0.0001 mg. (5 t.u.) is generally used. Regarding the patch test, I must say that although it is easy to do, unfortunately it is not a reliable test. In our recent study in the public schools in Houston, it was noted that the patch test missed about 50% of the Mantoux reactors, besides some false-positive reactions.

QUERY: Will you say something about the preparation of PPD—How long will the solution remain effective?

DR. HSU: Yes, you have brought up a very important point. The amount of tuberculous protein in PPD solution is extremely small. In preparing and drawing the solution, one would have to introduce some air into the vial. Therefore, air contamination is quite probable. If the solution is allowed to stand, and if it happens that some proteolytic bacteria were introduced into the solution, the potency of the solution will be greatly reduced. In order to be very sure of the potency of the tuberculin solution, one should prepare the solution fresh.

QUERY: Dr. Hsu, could you express your opinion as to whether we should treat a young adolescent who is a converter, perhaps not recent, one that you have had a negative tuberculin on a year before,

who now has a positive tuberculin and negative x-ray at the age of 12 or 13?

DR. HSU: Adolescence is a dangerous period for tuberculosis because there is a greater chance of developing progressive disease at this age. For this reason, special consideration should be given to these cases. I would advise giving INH therapy, especially if the tuberculin reaction is strongly positive.

QUERY: For one year?

DR. HSU: Yes, the treatment should be continued for at least one year as that is the minimal length for treatment considered adequate for tuberculosis.

QUERY: Dr. Hsu, in a recent article—it seems to be pretty authoritative—they refer to reading a tuberculin after 4 days and I wonder if that is a misprint. Will you talk about that?

DR. HSU: According to the diagnostic standard of the National Tuberculosis Association of the United States, the Mantoux test should be read between 48 and 72 hours, because after 72 hours the reaction starts to fade. Why this particular investigator prefers reading the test on the 4th day, I do not know. In a special recent project studying the duration of the tuberculin reactions, we have noticed that they fade considerably on the 4th day.

DR. SLOAN: I think there is some reason for the 4-day period in reading the patch test. The patch is left on 2 days, removed, and the test read 2 days later, which would be comparable to a 72 hour reading with a Mantoux test.

QUERY: Is there a possibility to reactivate the old lesion of the tuberculosis by doing repeatedly a Mantoux or PPD test?

DR. HSU: The dose of intermediate PPD is very small and the chance of such a small dose reactivating tuberculosis in children is very slight. That's why this intermediate strength has been recommended by the World Health Organization for screening tests. I am sure that children in many parts of the world are so heavily infected and many of them are very sensitive to tuberculin and yet this is the dose which has been used by the World Health Organization for screening. We have found at the Houston Children's Tuberculosis Clinic, that this dose is a safe dose to use, because we have not seen any untoward reaction such as fever or lymph node swelling following this dose of PPD.

QUERY: Is it possible to convert a negative reactor to a positive one by repeated tuberculin testing?

DR. HSU: That is a very pertinent question. This problem was considered when tuberculin was first prepared by Dr. Seibert. In her monograph on "The Chemistry of Tuberculin", she especially pointed out this problem. Because the tuberculous protein in PPD-S is heated protein, it is not very antigenic. The chance of atrificially sensitizing the patient is very, very slight with the small amount used in 5 t.u. PPD. We have been doing periodic tuberculin testing on the contacts of known cases of tuberculosis. Many children who have been tested 10 or 12 times still remain tuberculin negative.

QUERY: Do you see any of the chromogenic atypical acid fast tuberculous children in Houston and if so, how do you treat them?

DR. HSU: We have seen only a few cases in children as a I recall, they were cases involving the cervical lymph nodes which usually ruptured and drained. We have been treating them just like we treat tuberculosis and they have healed, but whether it was the treatment that brought about the good results or it was the spontaneous healing, we do not know. However, in the adults a large number of patients with photochromogen disease have been found. I think the latest count is 105. These are cases of pulmonary involvement. Many of them are advanced cases.

QUERY: The only organism?

DR. HSU: Yes. Atypical mycobacteria. Most of the cases were photochromogens. There were a few cases of scotochromogens and nonchromogens.

DR. SLOAN: I heard Dr. John Chapman in Dallas, who has a large experience with childhood T.B., speak at a meeting not long ago and he said that of the last 14 cases of cervical adenitis (scrofula) he had seen, only four were caused by mycobacterium tuberculosis; that of the other ten, eight were proved to be atypical acid-fast organisms and in two of them they could not determine the organism. The adenopathy in all 14 cases had the typical clinical and pathological characteristics of tuberculosis, so he thinks that a large number of cases diagnosed glandular tuberculosis in the Dallas area are caused by organisms other than mycobacterium tuberculosis.

QUERY: A word about hormones in the treatment of tuberculosis?

DR. HSU: You mean corticosteroids. This is cetainly a very frequently asked question. I really have no answer for it. I know the

Committee on Tuberculosis in children of the National Tuberculosis Association is planning on a nation-wide study just on this subject. We see a great deal of very enthusiastic reports on the use of corticosteroids as an adjuvant for treatment of tuberculosis, especially for tuberculous meningitis, tuberculous pleurisy, and miliary tuberculosis. I think in a child who is very sick we are justified to use everything which has been found to show beneficial effects by other investigators.

QUERY: Do you use steroids routinely in treating tuberculosis meningitis in Houston?

DR. HSU: We have not been using it routinely; however, we have tried it on several cases and we happened to get good results.

QUERY: Do you use steroids both intrathecally and systemically or just systemically?

DR. HSU: We use it intrathecally.

QUERY: What is the dose?

DR. HSU: We followed the dose recommended by Cocchi, an Italian investigator, starting with 2 mg. and then increasing the dose to 5 mg., giving it intrathecally, daily.

QUERY: Over how long a period?

DR. HSU: For 3 or 4 weeks.

QUERY: And in your severe tuberculosis meningitis, do you ever use steroids systemically?

DR. HSU: Yes, we use Meticorten, 1 mg./kilo per day. You may consider this a small dose. I must say our experience with tuberculous meningitis in Houston has been very limited because we have seen so very few cases of tuberculous meningitis since we started early treatment of primary tuberculosis in 1953.

QUERY: I would like to know what you would do with a patient who is about 9 months of age, who has a fairly strong positive patch test, but on two occasions a month apart had negative Mantoux tests, whose father and mother and other members of the family and baby also have negative chest x-rays, but whose father just recently quit working in a tuberculosis sanitorium? What would you do next with the baby—just continue to skin test the baby?

DR. HSU: If I had this child, I would repeat a tuberculin test on him. Since you have examined the contacts of this child and found

no source of infection, then we know at least, that this child is not in contact with tuberculosis in the home. However, since there was a positive patch test, I would follow through with intradermal tuberculin, using intermediate strength PPD.

QUERY: This is the first time I have ever had a child with a strongly positive patch test and negative skin test. Do you see them frequently?

DR. HSU: We have seen this kind of a case once in a while, that is, a child with a positive patch test and a negative intradermal test. We do not know exactly how to interpret that. I have asked Dr. Seibert. She said, "There is a possibility that the child is sensitive to something in the old tuberculin which is used in making the patch. In that case you would not get a positive tuberculin reaction with PPD. I would test the child with intermediate strength PPD for three consecutive months to be sure that his tuberculin reaction would not convert but remains negative".

QUERY: In the rare toxic reactions that you have had to Isoniazid, are any of them neurogenic?

DR. HSU: We have not seen any peripheral neuritis or convulsive disorders following the use of Isoniazid. Pyridoxine has not been used routinely for children since we have not seen any with peripheral neuritis even with high dosage of INH.

DR. SLOAN: I would like for you again to stress the dosage which I know sounds quite high to the individuals here who are treating adult tuberculosis. In other words, 20 to 30 mgs/kilo is the type of dosage we used in treating real severe tuberculosis. Isn't that true?

DR. HSU: Yes, for meningitis and miliary tuberculosis.

DR. SLOAN: And how about the child with segmental lesions and fever and also loss of weight—a good pulmonary case of tuberculosis—what dosage of Isoniazid do you use?

DR. HSU: We use 15 to 20 mgs/kilo per day along with PAS.

DR. SLOAN: Have there been in any parts of the world any higher dosages used in children than 30 mgs./kilo?

DR. HSU: I know it has been used as high as 50 mgs/kilo per day in some countries.

DR. SLOAN: In Puerto Rico?

DR. HSU: Yes.

DR. SLOAN: The representative from Puerto Rico at the meningitis prophylaxis study conference said that they quite often used

50 and occasionally 60 mgs./kilo in children in Puerto Rico. So, there must be a wide margin of safety in children. We have not here, to my knowledge seen any evidence of toxicity from Isoniazid and we use fairly high dosages.

QUERY: I would like to ask Dr. Hsu if in a dosage of streptomycin she has used, she has noticed any 8th nerve involvement. The Eli Lilly Company have just very recently withdrawn all of their Dihydrostreptomycins from the market due to its irreversible toxic effects on the nervous system. In the dosage used, how much involvement have you noticed with streptomycin?

DR. HSU: We have stopped using Dihydrostreptomycin in children since 1953 for fear of auditory nerve toxicity. Children are unable to recognize and complain of early hearing loss. Very often the hearing loss is not noticed until the destruction of the nerves is very far advanced. For this reason, we have avoided using Dihydrostreptomycin. Streptomycin at the dosage of 20 mgs/kilo per day appears to be safe. We have not seen toxicity such as vestibular disturbance nor deafness with exception of two cases. In these two cases, it was difficult to determine whether the cause of the deafness was meningitis or the drug.

QUERY: Although the use of B.C.G. probably masks one of the best ways of detecting tuberculosis in children, is its use indicated for people who are working with the disease, such as in the case of residents and nurses? Secondly, are such personnel that are positive tuberculin reactors immune, thus completely eliminating the indication for B.C.G.?

DR. HSU: I may quote what the United States Public Health Service recommends for the medical students and hospital personnel. These are the people whose exposure to tuberculosis cannot be avoided; therefore, B.C.G. vaccination is recommended. However, I have known of very, very few medical schools and hospitals in this country where routine B.C.G. vaccination has been given to the employees or to the students. For instance, at Baylor Medical School, there is not a B.C.G. program. Regarding the positive tuberculin reactors, B.C.G. has never been recommended for them

QUERY: I wonder if you would like to talk about the use of Isoniazid plus PAS, whether or not you get an emergence of resistance strains with the use of a single drug?

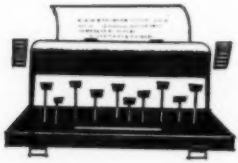
DR. HSU: The main reason for using PAS in combination with Isoniazid is to delay the emergence of resistance strains of tubercle

bacilli—I mean resistant to Isoniazid. Another reason is that concomitant administration of PAS raises the serum level of Isoniazid. Therefore, if we use Isoniazid alone the dose should be stepped up. If we use Isoniazid with PAS a smaller dose will be adequate. It must be pointed out that the emergence of drug resistant tubercle bacilli occurs mainly in patients with cavitary disease. In cases of minimal or no x-ray findings, INH alone without PAS, is now considered adequate therapy by many investigators.

DR. SLOAN: Any other questions? Dr. Blattner, do you have anything further?

DR. BLATTNER: No, except that I would like to emphasize that Dr. Hsu has taught us that the tuberculin test is a very difficult diagnostic procedure to do well. I think that we are thinking a lot more about tuberculins than we ever did before.

DR. SLOAN: Dr. Hsu, Dr. Blattner, it certainly has been a pleasure to have you with us and I think all of us have enjoyed it a great deal. If there are no other questions, the meeting is adjourned.



To the Editor
Archives of Pediatrics *July* 8, 1960
New York 3, New York

In my letter of September 24, 1959 which you published in the December 1959 issue of ARCHIVES OF PEDIATRICS, *it was suggested that chronic, low-grade or inapparent infection with Listeria monocytogenes might be the cause of some cases of habitual abortion. This was based on the reports of Gray et al.*[1,2] *who produced such a condition in rabbits; the observation of Seeliger*[3] *that 16 per cent of the women that had given birth to infants infected with L. monocytogenes had either aborted or given birth to stillborn infants during their previous pregnancies; and those of Rost* et al.[4] *who studied a series of women with histories of habitual abortion and found a sharp rise in antibody titer against L. monocytogenes at the time that pregnancy was interrupted. Similar observations were also made in other parts of Germany and Czechoslovakia.*[3,5] *Unfortunately, with the exception of the studies by Gray* et al.,[1,2] *the role of L. monocytogenes in these abortions could not be confirmed by isolation of the bacterium from either the aborted conceptus or the mother and were based entirely on serological findings. This letter is to call your readers' attention to the two papers published recently by Rabinovitz* et al.[6] *and Rappaport* et al.[7] *from the Hadassah Municipal Hospital, Tel Aviv, Israel reporting the isolation of L. monocytogenes from the cervical secretion of 25 out of 34 women with histories of repeated abortion. Eleven were pregnant at the time the isolation was made and three of these subsequently aborted. The remaining 8 together with 11 of the 14 non-pregnant women were treated with large doses of penicillin and sulphamethoxypyridazine (Lederkyn). As far as can be determined the pregnant patients are progressing normally. The number of previous abortions in these patients ranged from one to six and spanned a period of four months to 14 years. The number of positive cultures obtained from individual patients ranged from one to 15*

This department is for readers' views and observations. The editors will welcome reports or letters of pediatric and related-subject significance.

and in some the bacterium persisted for as long as 20 weeks. L. monocytogenes could not be isolated from 87 other women with no history of repeated abortion.

For the present it would be unwise to draw general conclusions from this study since it was confined to a relatively small geographical area where the general distribution of L. monocytogenes has received but little study. It has been isolated from animals in Israel on several occasions[8] but not previously from the human population. It was not stated whether these patients originated from rural or urban areas or if they were concentrated within a limited area where listeric infection might be endemic. However, if these data were typical for all areas, they imply that approximately 70 per cent of the women with histories of repeated abortion may suffer from inapparent listeric infection. Indeed, if this is further extended to include the 87 controls with no history of repeated abortion and from whom L. monocytogenes could not be isolated using the same culture techniques, it would indicate that almost 20 per cent of all women harbor the bacterium in their genital tract. If this were actually true, it would appear likely, especially in view of the extreme susceptibility of the uterine contents to listeric infection under experimental conditions,[1,2,9,10] that perinatal listeric infection would be much higher than available information indicates. It is quite probable, and this is supported experimentally,[2,9,10] that most infants who develop listeric meningitis during the first few months of life were actually infected prenatally as the result of undetected infection in the mother. Today it is known that women with bacteriologically confirmed low-grade listeric infection which is first manifested late in pregnancy can give birth to apparently normal healthy infants, providing the condition is diagnosed correctly and the proper therapy, usually a combination of tetracycline antibiotics and sulfa drugs, is initiated promptly.[3,11]

Since September, 1959, our laboratory in cooperation with the Communicable Disease Center of the U. S. Public Health Service is undertaking the serological typing of cultures of L. monocytogenes isolated from both human and animal sources. This program is supported by a National Institutes of Health Grant (E-2571) and its principal objectives are to stimulate a greater awareness of infections due to L. monocytogenes and to gain a better understanding of the epidemiology and pathogenesis of all forms of listeric infection. To date we have received 162 cultures from human sources, most of which have been isolated during the past two years and the majority during the past year. It is striking that of the total number, at least 56, or approximately 1/3, were isolated from infants under two years of age. Although an exact tabu-

lation is not presently available, a surprising number of the mothers of these infants have a history of abortion or other non-viable pregnancy. This finding coupled with those of numerous other investigators, and especially the recent papers of Rabinovitz et al.[6] *and* Rappaport et al.[7] *reemphasize that the possibility of infection with L. monocytogenes must be considered in all cases of repeated abortion and meningitis in infants. The true role of the bacterium in these syndromes can never be established without an awareness of this bacterium by both the medical profession and laboratory personnel who are alert to the difficulties which may be encountered in isolation attempts.*[12,13,14] *Our laboratory will gladly aid in the identification of gram positive bacteria isolated from these, or other conditions, and if it proves to be L. monocytogenes, determine its serological type.*

In spite of this accumulation of evidence incriminating L. monocytogenes as a cause of chronic infection in the female genital tract, there is considerable contrary evidence based on both naturally and artificially induced infections. Potel and Alex[15] *followed 19 women who had given birth to infected infants through 26 subsequent pregnancies. All but three terminated in the birth of an apparently normal healthy full-term infant. L. monocytogenes could not be isolated from the three non-viable pregnancies. Also it is well established that most animals that give birth to young infected with L. monocytogenes conceive normally and produce healthy young following rebreeding.*[1,2,9,10,16] *In view of this, a mother who has given birth to an infected infant need not necessarily fear a subsequent pregnancy. However, she and her obstetrician should be well aware of the potential hazards. Blood for culture and serological studies and cultures from the genital tract should be made at frequent intervals. The laboratory staff should be well aware of the possible difficulties of isolating the bacterium under these circumstances.*[12,13,14] *Appropriate therapy should be initiated immediately if there is a sudden rise in titer or marked change in the blood picture, or if any culture reveals the bacterium, or even in the absence of these, if the patient develops flu-like symptoms. With the proper precautions, such a pregnancy, in almost all instances, should terminate successfully. Regardless of the condition of the infant at birth, it should be observed closely for at least the first year for any sign suggestive of early meningitis.*

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